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**NF-kB induces lung maturation during mouse lung morphogenesis.**

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**Public Summary:**

In summary, we have developed an animal model that closely mimics the phenotypic changes that result in accelerated lung maturity secondary to an inflammatory stimulus at a critical period during gestation. This is a useful model to further study whether these early maturational effects might impact subsequent lung injury and repair mechanisms and whether they ultimately lead to a long-term impact on lung architecture and function.

**Scientific Abstract:**

Lung maturation is hallmarked by the appearance of surfactant-producing alveoli during transition from the saccular to alveolar stage of lung development. Inflammation can disrupt this process and accelerate lung maturity following intrauterine amniotic infection (chorioamnionitis). Nuclear factor kB (NF-kB) is a transcription factor central to multiple inflammatory and developmental pathways, including dorsal-ventral patterning in fruit flies, limb and mammary and submandibular gland development in mice, and branching morphogenesis in chick lungs. Given its shared role in inflammation and developmental signaling, we hypothesized that overexpression of NF-kB targeted to the lung epithelium would exert maturational effects on alveolar development. We generated transgenic mice with lung-specific overexpression of the RelA subunit of NF-kB using a surfactant protein C promoter construct. Our results showed that RelA overexpression in the lung yields increased alveolar type I and type II cells. These findings are consistent with a model whereby NF-kB may induce maturation of lung development through decreased apoptosis of epithelial cells.

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